THE

AMERICAN NATURALIST

VOL. XLIV *March, 1910* No. 519

THE IMPERFECTION OF DOMINANCE AND SOME OF ITS CONSEQUENCES[1]

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IT has long been recognized that in Mendelian heredity dominance is frequently imperfect. Mendel himself appreciated this fact, for he defines dominant characters as those "which are transmitted entire, or almost unchanged, in the hybridization" and points out that the hybrids between purple-red flowered and white flowered peas have lighter flowers than the darker parent. Indeed, in his letters to Nägeli he speaks of the *hybrid-form* of a character, recognizing that the hybrid character is not always that of one parent. Practically all hybridizers of the last nine years, and especially Correns and Bateson, have observed and laid emphasis on this imperfection of dominance. I can only add my testimony to theirs. It will be well to illustrate the general principle by some concrete examples. Bateson, and later Baur, found in hybrids between the prickly and non-prickly capsuled Datura that the prickles were much reduced in size. Correns found the hybrids between green- and yellow-leaved Mirabilis to have light green leaves; and in the same plant, variegation, though recessive, may be detected in the heterozygotes. Further, Mirabilis typica, 90 cm.

[1] A paper read before the American Society of Naturalists, December 29, 1909.

tall, crossed with *nana,* 40 cm. tall, produces offspring of 70 to 85 cm., thus failing to attain the dominant stature. Shull finds, in Shepard's purse, the heterozygote between elongated and non-elongated primary lobe to be imperfectly elongated, and the same investigator has devised a neat chemical experiment illustrating imperfect dominance. Morgan states that in hybrids between gray and albino rats, gray fails on the belly. In Lang's snails uniform shell color dominated over bands, but the hybrid shows pale bands. In poultry, extra toe dominates imperfectly over normal toe; complete inhibition of shank feathering dominates imperfectly over non-inhibition, so that some feathers appear; median comb dominates over absence so imperfectly that it is much reduced in the hybrids; dominant whites crossed with black give speckled females. Thus, the discoverer of dominance and his followers have generally recognized and laid emphasis on its imperfection; and were it not for certain careless exponents of Mendelism and a human tendency to substitute a skeleton formula for the living truth this introduction might have been spared.

Of imperfection of dominance there are all degrees, as Correns pointed out in 1905. The extreme case is complete failure to dominate; it has been observed many times. Thus Lock found that while black maize dominates over white, whites are in excess of expectation in subsequent generations and some of them prove to be heterozygous. Lang found hybrids between red and yellow snails to give exceptionally the recessive yellow. In poultry, median comb crossed with no median will give no median in 5 to 10 per cent. of the offspring. Extra toed crossed with normal poultry give over 20 per cent. of the recessive normal-toed type. The inhibitor of the narial flap when crossed with narrow nostril fails to activate in fully half of the offspring. Finally, cases are not unknown, as in certain rumpless fowl, when the dominant inhibitor has failed to act in the heterozygote completely. Several such cases have in recent years come to

light and it has been said that they show a *reversal* of dominance, *i. e.*, the normally recessive character has become dominant. Such an expression runs counter to the modern interpretation of dominance, which is that dominance depends on the presence of a determiner, recessiveness on its absence. Clearly a given character can hardly be now due to a determiner and again to its absence. The more reasonable hypothesis is that a determiner, though present, may fail to complete its ontogeny.

An insight into the cause of this failure is given by modern studies in cytology and breeding. These indicate that, ordinarily, in pure races, a well-developed, pure-bred character has a double determiner as its embryological anlage, while in the heterozygote the determiner is simplex. Now in just these cases when the anlage is simplex the character develops imperfectly; and it is not difficult to understand how, under certain circumstances, the simplex determiner might be insufficient for the development of the organ. Such would result in complete failure to dominate, not a reversal of dominance.

Further proof that dominance is not reversed, but only weakened, is derived from the facts of retardation in the ontogeny of some heterozygotes. For example, the white of the Leghorn is dominant over black, but, in the females at least, the dominance is so imperfect that the plumage of the hybrid shows many black spots or is spangled or it may be of a uniform blue; but in later molts it becomes pure or nearly pure white. Similarly, Lang found that the hybrid snails between the red and yellow forms sometimes showed at first the recessive yellow, but later gained the dominant red. The ontogeny of the heterozygote characteristic thus appears retarded and frequently fails altogether to reach its final goal.

This failure of the simplex determiner of the heterozygote to develop the corresponding characteristic is frequently found in one sex and not in the other. For example, the simplex determiner for horn production in sheep suffices to induce horns in the males but not in the

females. In the same way the simplex determiner induces color blindness in males but nothing less than the duplex determiner suffices to stimulate color blindness in females. One may say that, under the influence of testicular internal secretions the simplex determiner suffices for development, but not otherwise. No doubt the same is true for many other cases of "sex-limited heredity."

Imperfection of dominance goes even further. It is evinced not only in the heterozygote but even in pure-bred stock. This is the same as saying that, in breeding pure stock, a character may fail to develop even when a double determiner is present. Thus, in pure bred strains of Houdans or Dorkings having 5 toes on each foot, from 3 to 4 per cent. of the offspring fail to develop the extra toe on either foot. In certain races, at least, such as my Tosa fowl, two parents, both with complete inhibition of foot feathering, may have offspring in which this inhibitor fails to activate, so that their feet are slightly feathered. These cases of failure of a duplex determiner to work itself out in ontogeny are of the nature of sports—at least many sports are of the nature of defects arising in a pure-bred strain. In poultry one frequently gets such defects as failure of the neural tube to close (spina bifida), failure of the lower jaw to develop, absence of appendages and so on. In man, imbecility seems sometimes to occur as a sport; and it is doubtless a typical defect, transmitted as a recessive quality. It is probable that the same is true of hairlessness in mammals and man (Waldeyer, 1884, page 114, and Davenport, *Science,* November 20, 1908, p. 729). Since characteristics may occasionally fail of development in homozygotes their regular imperfection in heterozygotes is easier to understand.

Important consequences flow from the complete failure of a heterozygous characteristic. The first is, as pointed out by Shull, the difficulty of determining in the first hybrid generation what is recessive—since impotent dominance and recessiveness yield the same result. Indeed, one might be tempted to conclude with Shull that

when a character regularly fails to dominate it will never be practicable to distinguish the dominant and recessive conditions. But the situation is not so desperate as that—one clear difference between dominance and recessiveness remains, namely, while the dominant character, even when of duplex origin, sometimes develops fully, sometimes imperfectly, sometimes fails altogether; the homozygous recessive condition yields, on the other hand, offspring with entire absence of the quality. Since we can not know our homozygous recessives in advance the practical method of determining them is as follows: mate similars; some will yield families that show a great range of variation in the given characteristic from high development to complete absence, such are dominants or heterozygotes; others will yield families that show a limited range of conditions and will all be like the parents—such are homozygous recessives. Their progeny are uniform because absence of a character can not show a variability in ontogeny.

The foregoing principle may be illustrated by an example. In polydactyl strains no family (of over 2 individuals) from two syndactyl parents fails to produce some normal children, but several families from two non-syndactyl parents produce in a total of 119 offspring no syndactyl individual. The invariable families are certainly the product of two recessive parents.

The fact of imperfect dominance bears upon the controversy of alternative *versus* blending inheritance. When a booted fowl is mated to a clean-shanked one the offspring show grades of boot ranging from 0 to 9;—10 being the heaviest grade recognized. For example, when a Cochin is crossed with a White Leghorn the average grade of booting is about 4 on a scale of ten. Booting would seem to be a typical blending character. Yet the evidence of segregation is excellent. For, two extracted recessives (full-booted) beget no clean shanked offspring. $DR \times RR$ gives 50 per cent. of the offspring of grade 5 or over, $DR \times DR$ yields 25 per cent. of grade 5 or over,

while $DR \times DD$ gives none in these grades. A similar result is found in the apparently blending character of nostril height. May not segregation occur in the cases of apparent blending? Does not true blending occur only in complex characters such as stature and skeletal weight? Of such character complexes the component units may still segregate.

Finally, the fact of imperfect dominance leads to an explanation of many puzzling cases of apparent failure of inheritance. Some years ago I bought two tailless cocks *A* and *B*, of which *B* was said to be the son of *A*. They were certainly very similar in appearance. I mated *A* to some tailed hens and their offspring were tailed. The next year I mated these hybrids with each other and the females with their father. Were taillessness recessive, as I suspected, one-fourth of the progeny of the first mating and half of the progeny of the second should have been tailless. Actually there was produced not one tailless bird. One would apparently have been justified in concluding that taillessness is not an inheritable condition. But when the second tailless cock was mated with the tailless hybrids approximately half of the offspring were tailless; and such tailless offspring bred *inter se* have in turn produced a large proportion of tailless progeny. This whole case at first seemed inexplicable to me, as it did to Professor Bateson, to whom I related it, but it receives a satisfactory interpretation on the theory of imperfect dominance. For rumplessness, or rather an inhibitor of tail growth, is dominant over its absence. But with cock *A* this inhibitor is so impotent that in the heterozygote, at least, it does not make itself felt and even in the second hybrid generation the duplex determiner fails to activate fully. I say *fully*, for there was a trace of activity. At least fifteen per cent. of the offspring were recorded as having a small uropygium and in many of the adults the back appeared shortened and bent and the tail drooped instead of standing erect. Despite these evidences of the activity of the inhibitor, the

striking fact is that its activity is so feeble that it cannot prevent the development of the tail. In the case of the second cock, however, the inhibitor is stronger and behaves more nearly according to Mendelian expectation. Now, if a character can be so feeble as to fail completely in development in the heterozygote and even in the homozygote it will give the impression of non-inheritability; and I have little doubt that many cases in which there is apparently no or only a slight inheritance are due to a weak determiner. I could cite a considerable number of cases of this sort in my experience, but I refer for an account of them to a book that is being published for me by the Carnegie Institution of Washington. Still one other lesson may be drawn and that is the apparent variability in what I call the *potency* of determiners. The evidence for this potency seems to me to be strong, as it certainly is important for an interpretation of all non-Mendelian cases of heredity. By the aid of the facts of imperfection in dominance and the hypothesis of varying potency of determiners the territory to which the principle of the segregation of determiners is applicable becomes greatly extended.